Paper No. 27

THIS OPINION WAS NOT WRITTEN FOR PUBLICATION

The opinion in support of the decision being entered today (1) was not written for publication in a law journal and (2) is not binding precedent of the Board.

UNITED STATES PATENT AND TRADEMARK OFFICE

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BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES

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Ex parte MARK W. JACKWOOD
and
HYUK M. KWON

_____

Appeal No. 1996-1038
Application 07/922,492[1]

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ON BRIEF

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Before WILLIAM F. SMITH, ROBINSON, and LORIN, Administrative Patent Judges.

WILLIAM F. SMITH, Administrative Patent Judge.

DECISION ON APPEAL

This is an appeal under 35 U.S.C. § 134 from the final rejection of claims 7 through 11. Claims 25 through 31 are pending but have been withdrawn from consideration by the examiner.

[1] Application for patent filed July 30, 1992.

Claims 7, 8 and 10 are illustrative of the subject matter on appeal and read as follows:

7. A method of distinguishing between serotypes and/or detecting variants of infectious bronchitis virus comprising:

a. amplifying the S1 gene region of infectious bronchitis virus;

b. digesting the amplified S1 gene region with *Hae*III, or a restriction endonuclease which cleaves at the *Hae*III restriction site, to form a first set of restriction fragments;

c. digesting the amplified S1 gene region with *Xcm*I, or a restriction endonuclease which cleaves at the *Xcm*I restriction site, to form a second set of restriction fragments;

d. digesting the amplified S1 gene region with *Bst*YI, or a restriction endonuclease which cleaves at the *Bst*YI restriction site, to form a third set of restriction fragments;

e. separating the restriction fragments within the first, second and third sets by electrophoresis; and

f. analyzing restriction fragment length polymorphisms to distinguish between serotypes or detect variants of infectious bronchitis virus.

8. A primer for amplifying the S1 gene region of infectious bronchitis virus comprising a nucleic acid, having at least 20 nucleotides, which selectively hybridizes between nucleotide positions -100 through +1, relative to the ATG start site of S1, of the negative strand of infectious bronchitis virus.

10. A primer for amplifying the S1 gene region of infectious bronchitis virus comprising a nucleic acid, having at least 20 nucleotides, which selectively hybridizes between nucleotide positions 1600 through 1700, relative to the ATG start site of S1, of the positive strand of infectious bronchitis virus.

The references relied upon by the examiner are:

Lin, Z. et al. “A New Typing Method for the Avian Infectious Bronchitis Virus Using Polymerase Chain Reaction and Restriction Enzyme Fragment Length Polymorphism.” Archives of Virology, Vol. 116, (1991), pp. 19-31. (Lin)

Binns, M.M. et al. “Cloning and Sequencing of the Gene Encoding the Spike Protein of the Coronavirus IBV.” Journal of General Virology, Vol. 66, (1985), pp. 719-726. (Binns)

Claims 7 through 11 stand rejected under 35 U.S.C. § 103. As evidence of obviousness, the examiner relies upon Lin and Binns. We reverse the examiner’s rejection, and raise other issues for consideration by the examiner.

DISCUSSION

Lin discloses a typing method for infectious bronchitis virus (IBV) using polymerase chain reaction (PCR) amplification of the S2 gene and restriction fragment length polymorphism (RFLP) analysis of the amplified gene. Before conducting research on the S2 gene of IBV, Lin examined both the S1 and S2 regions of the gene. On pages 22-23, in the “Results” section, Lin states:

> The major antigenic determinant of IBV is the viral surface glycoprotein called spike (S) protein. S protein has been known to play an important role in virus neutralization.
>
> Therefore, we focused on the region of the S gene and first compared four S sequences. Figure 1 shows the comparison results, in which the number of nucleotides different from those present in the other three strains was scored for every 20 nucleotides. The nucleotide change at the N-terminal region of the S2 protein was relatively high in all four strains. In addition, this region is flanked by relatively unchanged conserved sequences. This region was thus selected as a PRC target.

From the comparison of the known sequences for S1 and S2, Lin concluded that the best region for the PCR/RFLP analysis was the N-terminus portion of S2 denoted in Figure 2 on page 22 since this portion exhibited the greatest nucleotide variation. Therefore, S2, and not S1, was chosen as the preferred site for the PCR/RFLP analysis.

The claims on appeal recite a method utilizing PCR/RFLP analysis of the S1 gene of the IBV using three particular restriction endonuclease enzymes. In our view, when Lin is viewed on its own, apart from appellants' disclosure of the present invention, there is no teaching or suggestion in Lin to utilize the S1 gene or the three recited restriction endonuclease (RE) enzymes in a method for detecting IBV serotypes. In this regard, we note that Lin uses nine specific RE enzymes for producing restriction fragments from the S2 gene of IBV. Nowhere does Lin teach or suggest which RE enzymes and how many would be successful in digesting the S1 gene to differentiate IBV.

Thus, although it was known in the prior art that the S1 gene of IBV is the target of neutralizing and hemagglutination-inhibiting monoclonal antibodies and that the S1 gene encodes the serotype specific neutralization epitopes, Lin chose not to analyze the S1 gene by PCR/RFLP analysis. Rather, after an analysis of both the S1 and S2 genes, Lin chose the S2 gene. It is only with the use of impermissible hindsight that Lin can be considered to teach or suggest the use of the S1 gene or the three claimed RE enzymes in

a method for distinguishing IBV serotypes. The secondary reference to Binns adds nothing more to the teaching of Lin since Binns merely discloses the nucleotide sequence of both the S1 and S2 genes of IBV.

In conclusion, we find no reasonable teaching or suggestion in either Lin or Binns concerning a method for distinguishing between serotypes of IBV by amplifying the S1 gene region of IBV and digesting the amplified gene regions with the three specifically recited RE enzymes. In addition, we find no reasonable teaching or suggestion in either Lin or Binns for the specific primers for amplifying the S1 gene region, as recited in claims 8 through 11, since Lin analyzes only the S2 gene of IBV and Binns merely sequences the entire S1 and S2 genes of IBV. For these reasons, we reverse the examiner's rejection under 35 U.S.C. § 103.

OTHER ISSUES

From a review of the application file, it does not appear that the examiner searched for all of the possible nucleotide sequences of the primers as recited in claims 8 through 11 since no documentation of a sequence search on these primers can be found in the administrative file. Upon return of the application, the examiner should ensure that all available electronic databases have been searched. In so doing, the examiner should recognize that the primer claims are "comprising" in nature and are,

thus, inclusive of relatively long nucleotide sequences. Such a search should be made of record in the application file.

REVERSED

William F. Smith Administrative Patent Judge	)
Douglas W. Robinson Administrative Patent Judge	) BOARD OF PATENT) APPEALS AND) INTERFERENCES
Hubert C. Lorin Administrative Patent Judge	)

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